BRITISH JOURNAL OF
TUBERCULOSIS
AND DISEASES OF THE CHEST

Vol. XLIV. April, 1950 No. 2.

GENERAL ARTICLES

PNEUMOPERITONEUM IN THE TREATMENT OF PULMONARY TUBERCULOSIS

By J. W. FRASER

From Glenlomond, by Kinross

Introduction

The possibility of using pneumoperitoneum in the treatment of pulmonary disease was first considered by Vajda in 1933, and in the following year Banyai (1934) reported on its use for the treatment of pulmonary tuberculosis. He demonstrated the possibilities of using an artificial pneumoperitoneum in combination with phrenic nerve block in order to raise the diaphragm, and so create an " air splint " below the diaphragm when collapse therapy could not be carried out by other means. Banyai also mentioned that pneumoperitoneum had led to symptomatic improvement in intestinal tuberculosis among forty-four patients with advanced pulmonary tuberculosis. Thereafter he treated another 100 cases, and " in many cases symptomatic relief was so striking that a more extensive use of pneumoperitoneum has been carried out since." Fremmel in 1937 accidentally induced pneumoperitoneum in two patients having artificial pneumothoraces induced through the lower intercostal spaces in the mid-axillary line. After twenty-four refills one of the patients had an established pneumoperitoneum; both patients did well. Fremmel recommended pneumoperitoneum for pulmonary tuberculosis except for " chronic indurated tuberculosis of several years' standing where the influence of pneumoperitoneum would probably be negligible."

Trimble and Wardrip (1948) have pointed out that with all the available collapse measures (from pneumothorax to thoracoplasty) " there is still a large group of patients for whom we have had little to offer in the way of an active approach to their therapeutic problem." These authors were favourably impressed by the use of pneumoperitoneum on eighty cases, although it was used where there was practically nothing else to offer in the way of treatment. Failure with many artificial pneumothoraces due to limiting adhesions was mentioned by Joannides and Schlack (1937), and they recommended the use of a paralysed diaphragm and pneumoperitoneum. Trimble, Eaton and Moore (1939) also recommended pneumoperitoneum when artificial pneumo-

thorax was ineffective " either because of inability to find a free pleural space or because only a small ineffective collapse could be obtained," particularly when more drastic types of collapse therapy were not indicated at the time. They further advocated pneumoperitoneum for that large group of patients with extensive or advanced bilateral lesions still in the exudative stage, on whom other forms of collapse therapy were neither practicable nor safe. Rilance and Warring (1941) considered pneumoperitoneum as a procedure of merit. Fowler (1941) thought its value due to its ability to improve the patient's general condition or alter the course of the pathological process so that a more direct approach by radical surgery became safer. Clifford-Jones and Macdonald (1943) and Edwards and Logan (1945) also believed that pneumoperitoneum had a place in collapse therapy. From India, Mallick, Malhotra and Mohammad (1943) also reported favourably on its use, and other support has come from Drury and Duffy (1944), Keers (1948), Gilmore (1947), and Whitehead (1948).

In the past few years various American authors have amassed impressive figures from which estimates may tentatively be made on the value of pneumoperitoneum, and in this country Hounslow (quoted by Kayne, Pagel and O'Shaughnessy) has a considerable total of treated patients since 1941. The remarks of some of those authors are illuminating. Thus Crow and Welchel (1945) stated that " phrenic nerve interruption and pneumoperitoneum can be effective in a much larger number of patients than any other method of collapse therapy," and they believed that pneumoperitoneum will mark a much greater advance in the treatment of pulmonary tuberculosis than did the beginning of the extensive employment of artificial pneumothorax. Anderson and Winn (1945) considered their results had been remarkable in treating 110 negroes, and believed that for suitable cases " the results will be as good as or better than those seen with artificial pneumothorax."

It is thus evident that a number of authors have been favourably impressed by the therapeutic possibilities of pneumoperitoneum in pulmonary tuberculosis. There have been, however, many conflicting opinions, inevitable with a new method of treatment. Thus it is only during the last twenty years that the definitive indications for the extensive use of artificial pneumothorax have been evolved, despite the fact that Forlanini published his original paper in 1895, and his results from the more extended use of artificial pneumothorax in 1906. Some institutions make use of pneumoperitoneum either in small or large measures; others find it of use for a small number of patients only; others again apparently have never practised this method of treatment at all. In 1943 Drolet quoted various writers to the effect that pneumoperitoneum was not very enthusiastically received. As Drolet's article referred to the period 1937-41 his comments are not surprising. It is only during the past few years that pneumoperitoneum has become an accepted form of collapse therapy in this country. Hounslow's figures date back to only 1941, when pneumoperitoneum was originally used at Clare Hall Hospital, and its first mention in the statistics of the Brompton Hospital Reports was made only in 1944. Nevertheless, Rafferty (1945) and Pinner were not impressed by pneumoperitoneum, and in 1948 Howlett wrote: " It does appear that great divergence of opinion continues to exist in spite of the fact that well over a decade has

passed since the procedure was first recommended." He went on to state that until recently it had been extremely difficult to judge to what extent this sporadic but spreading enthusiasm for pneumoperitoneum is justified, as few large enough groups of patients have been reviewed, and the follow-up was generally too brief to permit mature judgment. But having decided that there was indeed some virtue in pneumoperitoneum, he believed that it still had to establish a claim to the extensive and enthusiastic use accorded in certain sanatoria and clinics. Some of the results reported, however, do go some way to establish pneumoperitoneum as a successful procedure. Thus in Crow and Whelchel's series cavity closure was obtained in 63·3 per cent. of 546 patients. In Anderson and Winn's 110 negro patients there was cavity closure in 58·4 per cent. Mitchell *et al.* gave similar encouraging figures, for they found 57 per cent. results satisfactory in 188 white patients, and 37 per cent. in 286 coloured patients with advanced pulmonary tuberculosis not amenable to conventional forms of collapse therapy. Trimble *et al.* (1939) gave similar figures; 57 per cent. of their patients achieved arrested disease and another 13 per cent. were improved. Hounslow's figures showed that 83 per cent. reached quiescence or were improved. It is believed from those results that pneumoperitoneum has indeed something to offer as a form of collapse therapy.

Technique and Management

The site of injection is largely influenced by the operator's own preference, although a majority favour the left sub-costal route. Banyai, however, actually prefers the sub-phrenic route through one of the intercostal spaces on the right. But in this there is a very real danger of inducing an artificial pneumothorax, as reported by Fremmel (1937) and Gaetan (1939). The actual technique of induction is widely enough known to make description mere repetition. Certain points, however, should be emphasised. Thus there is little danger in traumatising the bowel, as it appears to be pushed forward by the advancing needle—always provided, of course, that the needle is not violently pushed into the abdomen. But trauma to the bowel has been noted. To avoid it all injections should be made remote from the site of any abdominal operation scars, to which bowel might possibly be adherent. In contradistinction to artificial pneumothorax there are no manometric readings to indicate that the needle has indeed entered the peritoneal cavity. The intra-abdominal pressure is initially atmospheric, and thus there is no free swing on the manometer. No matter how often a pneumoperitoneum is induced the added precaution is always taken of frequently turning off the apparatus in order to read the manometer which registers as soon as air has entered the peritoneal cavity. Provided the needle has been correctly introduced a free flow of air into the peritoneal cavity will not cause a large positive pressure.

Various suggestions have been made, notably by Banyai, that patients should be treated in the semi-recumbent position, or, according to others, with the patient flat, and with the side of the paralysed diaphragm uppermost to allow the air to rise further on that particular side, with the possible action of raising still further the paralysed diaphragm. This thesis never seemed very sound from the available evidence. Now, Howlett (1948) quotes Wright to the effect that when a patient changes from the erect to the recumbent attitude, the

lung volume at mid-capacity (*i.e.*, the air content of the lung at the end of an ordinary quiet respiration) is reduced by 25 to 50 per cent. With a pneumoperitoneum somewhat similar findings were obtained, and with the addition of phrenic paralysis (on two patients only), there was further decreased volume to a marked degree (50 per cent. over the expected level in both positions). Thus there would appear to be sound physiological reasons for bed rest in promoting the relaxation so important for healing diseased tissues. Most patients in this hospital are kept several months on bed rest, and only when there is clinical and X-ray evidence of satisfactory progress are they allowed up.

The use of an abdominal binder has been recommended by some writers, and not really thought necessary as a routine by others. Banyai considered that it " increases the rise of the diaphragm in addition to that caused by any given amount of air." However, we do not consider a binder necessary except to support the loose abdominal walls of parous women; those patients can be helped by a well-fitting corset. In most patients the good tone of their abdominal muscles is adequate to maintain a high diaphragm. Hawkins and Thomas (1946) agree with this.

Duration of Pneumoperitoneum

At the present time no adequate number of patients has been followed long enough for any estimates to be made on how long pneumoperitoneum should be continued. Thus Mitchell *et al.*, up to 1946, had forty-two patients who had received treatment for two years, fourteen over three years, eight over four years and four over five years. Trimble *et al.* could present only forty-one arrested patients up to 1946, in whom therapy had been voluntarily discontinued after a period of three and a half years. From these small numbers it is not possible to say how long treatment should continue. But it is difficult to imagine that the duration of pneumoperitoneum should be very much different from that of artificial pneumothorax. References by Rafferty (1945) on the duration of collapse in artificial pneumothorax vary between three and four and a half years, and these are selected groups of patients who had survived the numerous hazards. In general pneumoperitoneum therapy, as the sole procedure, should last probably for not less than three years. When other collapse measures are indicated pneumoperitoneum should then last for a much shorter time, depending on the clinical course. Edwards and Logan summed it up: " Where a lesion can be controlled by pneumoperitoneum and by no other method, it will be necessary to maintain treatment for as long as artificial pneumothorax."

Complications

A variety of complications are met with in pneumoperitoneum therapy in the same way as these are encountered in artificial pneumothorax, but they are not so serious nor so crippling to the patient, and it has been stated: " Compared with pneumothorax therapy, we have no hesitation whatever is asserting that the inherent risks are quite strikingly less frequent in materialising and less formidable in degree " (Kayne *et al.*, 1949). Accidents, of course, have occurred, but with a more thorough appreciation of technique, and

probably with a little added care, the number of really serious accidents appears to be negligible.

In reported series the more important complications were air embolism, mediastinal emphysema, and spontaneous pneumothorax, accidental pneumothorax, peritoneal effusion, tuberculous peritonitis, obliterative peritonitis, perforation of the bowel, and various abdominal herniæ.

(1) *Air embolism* may be transient or fatal; deaths from this cause have been reported by Aslett and Jarman (1945), Simmonds (1946) and Bailey (1948). Death has also been reported from trauma to a large omental vein, although embolism was not the cause (Cornwall and Partridge, 1947).

(2) *Mediastinal emphysema* has been described by Simmonds, and one patient also developed a *spontaneous pneumothorax*.

(3) *Accidental pneumothorax* can occur by giving pneumoperitoneum refills through the lower intercostal space.

(4) *Peritoneal effusion* is by far the most important complication encountered in pneumoperitoneum therapy; its frequency varies in published reports, but seems to vary between 2 and 8 per cent. of all cases. In simple effusions of small quantity, some authors recommend continuing the refills, as the fluid will usually be absorbed within weeks or months. Trimble *et al.* (1939) have examined the peritoneum in patients who had had pneumoperitoneum, and who eventually came to autopsy; the local effects of air in the peritoneal cavity had been quite benign, and compared favourably with local effects of air in the pleural cavity. Ascites was observed, but did not require any treatment. Even when present in large amounts, it was not necessarily indicative of an inflammatory reaction in the peritoneum.

In the present series five cases (5 per cent.) which had peritoneal effusion were seen; in all cases the pneumoperitoneum was abandoned, as it was not considered advisable to continue refills in the presence of fairly large ascites. All these patients were completely asymptomatic, the fluid levels being noted on routine screening. In no case was aspiration necessary.

(5) *Tuberculous peritonitis* has been reported in approximately 3·5 per cent. of patients. These cases with this complication have been clinically ill, and it necessitated termination of the pneumoperitoneum. It has been suggested that the tuberculous peritonitis pre-exists, possibly in the majority of such cases (Anderson and Winn). " The probability is that tuberculous peritonitis will occur no oftener with pneumoperitoneum than it will in the tuberculous patient without pneumoperitoneum therapy " (Trimble *et al.*, 1939). This is probably an accurate assessment of its frequency.

Three cases (3 per cent.) were found in this series, and in two of those it was doubtful whether the pneumoperitoneum had anything to do with its occurrence. One of those patients had continued activity of the right lung disease with a positive sputum; she later developed a left pleural effusion. Another patient had a tuberculous consolidation of the right lung with positive sputum; he developed a large gland mass in the ileo-cæcal region, and with it a tuberculous peritonitis. The third patient died at home with what was described as peritoneal effusion; no other particulars are available.

(6) *Peritoneal adhesions* are seen in the majority of patients with pneumoperitoneum (Anderson and Winn quote 70 per cent.), but these cause no

difficulty. However, in a certain number there is a progressive obliterative peritonitis which makes refills increasingly more difficult, finally leading to abandonment of the pneumoperitoneum. Two such cases were seen in this series, one of whom had severe abdominal pain with refills.

(7) *Perforation of the bowel* has been reported (Clifford-Jones and Macdonald, 1943).

(8) *Abdominal herniæ* have been variously reported, but these can be invariably controlled by a properly fitting truss. A series of recent reports has appeared giving accounts of scrotal pneumocele. All reports agree that this complication is due to air herniating in the potential space of an obliterated tunica vaginalis.

In this series no gross abdominal herniæ were seen. Several patients had small umbilical herniæ causing no upset nor any discomfort.

Among other complications is pleural effusion; in this series it was seen in one patient who had already had tuberculous peritonitis. Pleural effusion should probably not be considered as a complication of pneumoperitoneum, as it was found no oftener than among any tuberculous patients.

A much more serious complication was seen in a patient who developed a lobar atelectasis; this patient was known to have had tracheo-bronchial tuberculosis. Similar findings were noted by Brian and Ricen (1941).

TABLE I.—LIST OF COMPLICATIONS ENCOUNTERED IN PNEUMOPERITONEUM TREATMENT (100 CASES)

Peritoneal effusions	5
Tuberculous peritonitis	3
Obliterative peritonitis	2
Lobar atelectasis	1

Table I gives a list of the complications encountered in this series; these major complications all necessitated termination of the pneumoperitoneum. They amount to 11 per cent. of the total cases and compare with Trimble *et al.* 6·7 per cent., and Mitchell *et al.* 4·7 per cent.

Indications

Broadly speaking, two groups of patients have been recommended for pneumoperitoneum—the "failed artificial pneumothorax" group and the far advanced. In many of the latter pneumoperitoneum has probably been given more as a solace to the physician's conscience, and to raise patients' morale, rather than with any more precise hope of reaching a better result. Considering the comparative lack of complications, pneumoperitoneum is always something that can be tried.

(1) *Unilateral tuberculosis where an artificial pneumothorax has failed,* and the next step would formerly have been a thoracoplasty. Pneumoperitoneum should be tried as an intermediate procedure. The intermediate stage of pneumoperitoneum may in some cases give a satisfactory result obviating the use of major surgery.

(2) *Unilateral tuberculosis where pneumoperitoneum is used with artificial pneumothorax.* There may be insufficient relaxation of the collapsed lung in artificial pneumothorax by reason of apical or diaphragmatic adhesions; the addition of pneumoperitoneum may then be sufficient to lead to cavity closure.

(3) *Predominantly unilateral disease where pneumoperitoneum is used as a primary procedure.* This use is to help a patient to stabilise acute exudative disease before using other collapse measures, either artificial pneumothorax or thoracoplasty. Although acute exudative disease is the predominant indication, more chronic lesions have also been successfully treated by pneumoperitoneum, though there is then less certainty of a successful end-result.

(4) *Bilateral disease* which is either too acute or too advanced for any other immediate form of therapy. The patients in this category might be classified as follows:

(*a*) Those on whom pneumoperitoneum is tried in combination with artificial pneumothorax on the other side.

(*b*) Those on whom, if one side is improved sufficiently, major surgery might be considered on the other.

(*c*) Those on whom pneumoperitoneum is indicated as a primary procedure. This is a large group for which little or no alternative active treatment has till recently been available. Pneumoperitoneum can sometimes be used for those patients in the hope that it might help.

(5) *To close empyema spaces*, facilitate absorption of fluid and help to re-expand " unexpandable lungs."

(6) *To control severe pulmonary hæmorrhage*, pneumoperitoneum has been recommended by Benatt (1946) and by Logie *et al.* (1943). It can be used where the bleeding cannot be controlled by pneumothorax, or where the source of the bleeding is not known in bilateral disease.

Contra-indications

Contra-indications are few, and do not cover so wide a field as those for artificial pneumothorax; indeed, contra-indications for pneumothorax become, in many cases, an indication for pneumoperitoneum. Many contra-indications mentioned in the literature are not really contra-indications to pneumoperitoneum as such, but rather to active intervention of any kind. Thus generalised tuberculosis and amyloidosis have been mentioned, and moribund patients with extreme toxicity, where there is little point in using any collapse therapy unless the patient shows signs of resisting the disease successfully by himself. Far advanced disease with large thick-walled cavities will resist collapse by pneumoperitoneum, just as it will any other form of collapse therapy. It has also been suggested that serious heart disease is a contra-indication; thus Banyai states that coronary disease and cardiac decompensation should be considered contra-indications. One can only agree, for there appears to be little point in instituting any form of collapse therapy in the presence of marked cardiac disease. In this connection it might be added that Benatt and Berg (1945) found that pneumoperitoneum did not cause any electro-cardiographic changes in the normal individual.

Certain abdominal factors are contra-indications to pneumoperitoneum, these are:

(1) Limited diaphragmatic movement, as seen on screening—*e.g.*, patients with old-standing pleurisies or empyemas, or who have extensive

abdominal adhesions to limit diaphragmatic excursions. In the analysis of results it will be seen that in no patient with an empyema or pleural effusion was a satisfactory rise of the diaphragm obtained.

(2) Presence of severe abdominal disease—tuberculous gland masses and large tumours.

In this series of patients no contra-indication to pneumoperitoneum was seen except one: the complications associated with tracheo-bronchial tuberculosis—viz., tension cavities, lobar and pulmonary collapse. Tracheo-bronchial tuberculosis is always a major problem in collapse therapy due to interference with bronchial drainage. It has been reported that pulmonary tuberculosis is associated with tuberculosis of the main bronchi in 10-15 per cent. of patients. Rafferty gave the figure 11 per cent. and Salkin *et al.* (1943) 15 per cent., a figure rising to 40 per cent. at post-mortem examination. Samson (1937) considered that collapse therapy should not be given, and pointed out that the results were equally poor with rest only or collapse therapy. He also pointed out that tuberculosis of the bronchi may be seen only after collapse measures are instituted. In this connection Warren *et al.* (1938) recommended that all patients should be bronchoscoped if there was any doubt about the presence of tuberculosis of the bronchi. Many other authors agree with this, but it is an ideal not yet generally available to most patients in this country.

Trimble *et al.* used pneumoperitoneum therapy in the known presence of tuberculous bronchitis in seventy-four patients. The results were not good, but compared with any other treatment available the results in the minimal and moderately advanced cases were not unsatisfactory. Their work antedated the use of streptomycin, though how far this is itself satisfactory yet remains to be seen.

In the present series four patients had clinical and radiological evidence of tuberculous involvement of the bronchi, though if routine bronchoscopy had been done no doubt many more examples would have been discovered. Three of the four patients developed typical tension cavities, eventually requiring abandonment of the pneumoperitoneum. Another patient had a lobar atelectasis, and later a complete lung collapse; bronchoscopy showed tuberculous granulation tissue blocking the right main bronchus. It would seem, then, that where there is any suspicion of tracheo-bronchial tuberculosis, bronchoscopic examination should be done before starting any form of collapse therapy. Where it is found streptomycin should be tried first and may then well make collapse therapy possible later on.

Results

In this series the results of 100 consecutive patients submitted to pneumoperitoneum are given. The age, distribution and classification by sex are given in Table II.

The results have been considered according to whether they were successful or unsuccessful or whether the patients died. The successful group has been further subdivided into satisfactory and improved. By "satisfactory" is meant an individual with an apparently controlled lesion both clinically and radiologically, and who had a negative sputum either on culture or after

PLATE II.

Case 1.
Male,
Age 26.

Fig. 2—4:3:49.

Fig. 1—6:2:47

To face page 38

PLATE II.

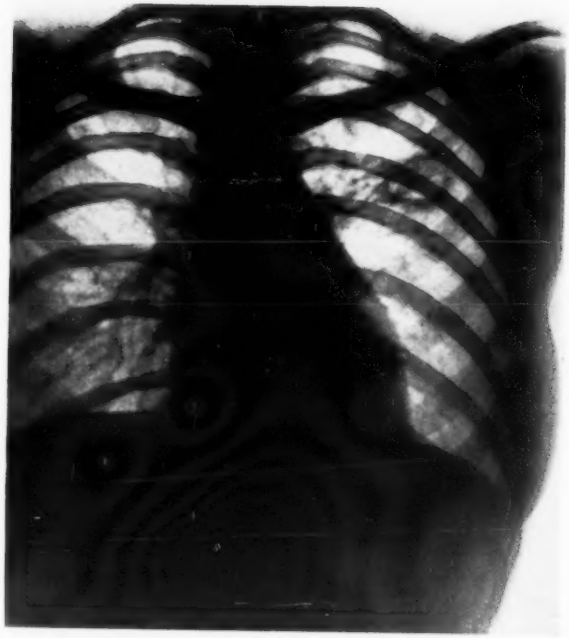
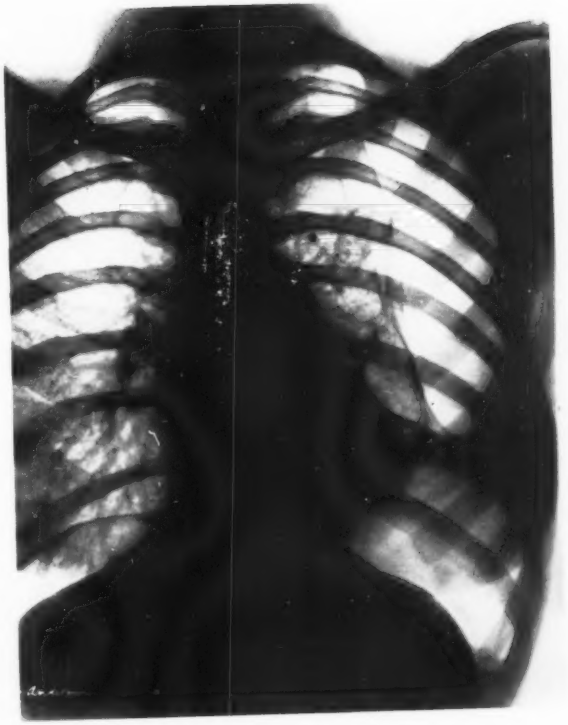


FIG. 3—18:12:46.

CASE 2.
FEMALE,
AGE 28.

FIG. 4—10:9:47.

PLATE III.



CASE 2 (continued). FEMALE, AGE 28.
FIG. 5—17:9:48.

To face page 39

PLATE IV.

FIG. 6—8:5:47.

CASE 3.
FEMALE,
AGE 15.

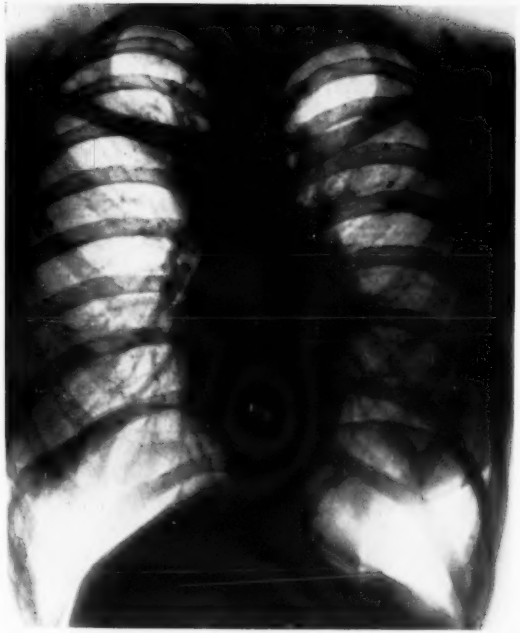


FIG. 7—4:2:47.

TABLE II.

	Males			*Females*		
Age	*Min.**	*M.A.†*	*F.A.‡*	*Min.**	*M.A.†*	*F.A.‡*
10-19		3	3	1	4	5
20-29		11	12		16	15
30-39	1	5	7		2	5
40-49	1	2	4		1	1
50-over						1
Totals ..	2	21	26	1	23	27

gastric lavage and guinea-pig inoculation. In the improved group are patients (1) who had improved from a pneumoperitoneum as the sole measure and where the disease appeared to be controlled, except that a positive sputum could still be obtained by culture or gastric lavage; or (2) who improved sufficiently in the presence of extensive disease to make other collapse measures possible, but in whom there was not satisfactory evidence of controlled lesions, the sputum remaining positive.

Table III shows the results according to age.

TABLE III.

Age	*Satisfactory*	*Improved*	*Unsuccessful*	*Died*
10-19	2	6	10	
20-29	6	10	30	4
30-39		4	14	3
40-49	4	1	4	1
50-over			1	
	12	21	59	8

It will be seen that 33 patients had a successful result—*i.e.*, 33 per cent. of all patients were benefited completely or in part during pneumoperitoneum treatment. Of these 33 patients, 15 (34 per cent.) of the moderately advanced and 17 (32 per cent.) of the far advanced cases had a successful result. Considering that little prospect of alternative treatment could be offered to many of them, these results can fairly be regarded as favourable. Too few patients have had pneumoperitoneum terminated for a sufficiently long period to make any definite comment on end results.

Table IV shows the distribution of the lesions according to the zones predominantly involved and the results obtained. In many it should be understood that more than one zone of lung was involved; there was extensive bilateral disease in many.

* Minimal disease. † Moderately advanced disease. ‡ Far advanced disease.

TABLE IV.

	U.Z.	*M.Z.*	*L.Z.*	*Pleura.*
Satisfactory	7	3	3	1
Improved	16	2	1	
Unsuccessful	28	15	11	5
Dead	7	1		
Total	58	21	15	6

Of all the patients in the entire series 35 had upper lobe cavities on the right and 15 on the left; 17 successful results were seen on the right and 7 on the left. It has been shown by Ellison and Tittle (1943) that a marked rise on the diaphragm on the right side frequently leads to a kinking of the eparterial bronchus, resulting in atelectasis of the right upper lobe giving cavity closure.

Cavities lying in the middle zone are, in many cases, hilar cavities, and as such are usually occupying the apex of the lower lobe and lying in the paravertebral gutter. The area is often widely adherent, preventing the diseased zone from collapsing. Ellison and Tittle have also pointed out how cavities lying towards the base may be entirely unaffected by a risen diaphragm. They accounted for this as due to fixation of the attachments of the diaphragm so that stretching of the dome of the diaphragm upwards only resulted in the normal triangular space behind the dome of the diaphragm becoming exaggerated. If the lung is adherent in the costophrenic angle there is no possibility of any rise of the diaphragm to obliterate this triangular space with respiration. This fact has been demonstrated radiologically by Morgenstern and Pine (1948).

In the overall results it is seen that 40 per cent. of upper zone lesions, 24 per cent. middle zone, and 26 per cent. lower zone lesions gave successful results. Thus upper zone lesions appeared to benefit much more often than with those in other zones. Contrary to some authors, it is not considered that lower zone lesions are a primary indication for pneumoperitoneum. Lesions in this situation always present great difficulty, for thoracoplasty means a great loss of normally functioning lung tissue. Neither has artificial pneumothorax been entirely successful, for many of these lower lobes show an absorption collapse after pneumothorax has been induced, indicative in many cases of tuberculous changes in the bronchi. It has been pointed out that this is three times as common in the lower lobe as in the upper (Salkin *et al.*, 1943). Two patients in this series showed absorption collapse of their left lower lobes after the induction of artificial pneumothorax.

In the pleural group only one patient had a successful result; a patient who had pneumoperitoneum induced at the onset of an effusion into an artificial pneumothorax. The others were quite unaffected by pneumoperitoneum. In all of them fluid or pus had been present for several months before pneumoperitoneum was instituted; by that time the diaphragm was firmly fixed by adhesions. To be effective pneumoperitoneum needs to be started directly

fluid is observed in an artificial pneumothorax, so that thickened visceral and parietal pleuræ do not limit its help. Nevertheless several other recent cases with pleural effusions (not in this series) who had pneumoperitoneum induced at the onset of the effusions did not show any successful rise of the diaphragm on the affected side.

Discussion

For any form of collapse therapy the end results alone will provide a basis on which to determine its efficacy. But as so few patients have had pneumoperitoneum terminated for a long enough period, comparison with other methods of therapy is not yet possible. The limiting factors with artificial pneumothorax are now better understood than formerly. Realisation of the bad end results associated with pneumothorax complicated by fluid or pus has in turn led to other forms of therapy. A satisfactory artificial pneumothorax is probably initiated in only some 10 per cent. of all patients; those patients still have to undergo the hazards of its course. In others with adhesions there are the risks of adhesion section, loss of the pneumothorax by reason of effusion, blood (5 per cent.), or empyema (10 per cent.) (Watt, 1947). Since all these complications are periodically encountered after thoracoscopy one always wonders whether pneumoperitoneum as an initial procedure might not eventually have produced a much more satisfactory result. It has already been shown that pneumoperitoneum is associated with few serious complications and, as a form of treatment, is as easily managed as (probably more so than) artificial pneumothorax. Moreover, it is not associated with any disturbance of the pleura to lead to effusions and empyema.

In this series it appears to have justified its place as a form of collapse therapy by reason of the good results obtained. Pneumoperitoneum has improved several patients quite remarkably; others have been helped towards thoracoplasty, which has been less extensive in many than had originally seemed possible. Though it may only serve as an intermediate stage in treatment, one of its main indications is the aim of improving a patient's general condition so that further collapse measures can be used with a greater hope of success. In some the end result of pneumoperitoneum was much better than expected, and in several thoracoplasty was avoided.

Some authors (Rafferty, 1945; Shipman, 1942) have discussed the use of primary thoracoplasty on the ground that it is questionable whether the routine use of (or attempt at) artificial pneumothorax in all cases requiring major collapse therapy is justified. From the results obtained in this series it seems that a routine use of pneumoperitoneum prior to thoracoplasty, except in thick-walled cavities, should be considered. It is, however, really the acute exudative lesions that show the most dramatic results with pneumoperitoneum. It is precisely with such acute lesions that artificial pneumothorax is contraindicated, since they are so frequently complicated by effusions and empyema. Numerous authors have commented on the adverse effect of an artificial pneumothorax complicated by effusion, and the shrunken hemithorax following an empyema is to be avoided at all costs. There is an impairment of pulmonary function following uncomplicated pneumothorax (Birath, 1947), and more markedly so in the presence of pleural effusions. This end result,

in so far as is at present known, does not occur with pneumoperitoneum. Large thick-walled cavities are not likely to respond to pneumoperitoneum; Potter (1941) has pointed out that these cavities resist even manual compression at post-mortem. Large apical cavities, cavities lying under a thickened visceral pleura with an adherent lung, or peripherally located cavities with widespread adhesions, are not likely to respond to pneumoperitoneum. Tension cavities due to bronchial stenosis are not successfully treated by pneumoperitoneum.

It cannot be doubted that cavity closure can occur with bed rest as the sole measure; those cases have been described (Fales and Beaudet, 1934; Grenville-Mathers, 1947), and most tuberculosis physicians have seen it occur. Pneumoperitoneum, however, does seem to speed up the process, and gives a higher percentage of successful cavity closures. It is not intended that this should be done in the absence of bed rest; bed rest and pneumoperitoneum are not two competing forms of therapy; each is necessary to the other and both are essential in the same programme.

Summary

1. Pneumoperitoneum is a relatively simple procedure to use, and there are few complications or contra-indications.

2. The indications for its use are reviewed.

3. The results in 100 patients submitted to pneumoperitoneum show that 33 per cent. gave a successful result. Forty per cent. of upper zone lesions, 24 per cent. of middle zone and 26 per cent. of lower zone lesions gave successful results.

4. Pneumoperitoneum is considered to be a procedure of value and should have a place in any collapse therapy programme.

APPENDIX

Case 1; Male, age 26.—Admitted to hospital on 27.1.47 with acute disease in upper half of right lung and light infiltration in lower half of left lung (Fig. 1). On 7.3.47 a pneumoperitoneum was induced without any phrenic paralysis. There was collapse of the right upper lobe against the mediastinum and the present position is seen in Fig. 2 (4.3.49).

Case 2; Female, age 28.—X-ray on 18.12.46 (Fig. 3) on admission showed a moderately large cavity in the left infra-clavicular area. On 19.12.46 a left artificial pneumothorax was induced, but was not really satisfactory despite two attempts at adhesion section; a mid-zone cavity persisted in the collapsed lung. Left phrenic nerve was crushed on 27.5.47, but X-ray (10.9.47) (Fig. 4) showed that the cavity persisted. On 23.9.47 pneumoperitoneum was induced; the pneumoperitoneum and artificial pneumothorax were used simultaneously to obtain cavity closure, as is seen in Fig. 5. This patient has terminated all treatment, she has cavity closure and is sputum free.

Case 3; Female, age 15.—Admitted on 6.5.47 with acute disease of right lung and of left mid-zone (Fig. 6). The right phrenic nerve was crushed on 24.6.47, and pneumoperitoneum was induced on 8.7.47: the right diaphragm quickly recovered function and thereafter the left diaphragm was paralysed on 23.9.47. This patient's condition improved considerably. She appeared to have cavity closure on the right, but signs persisted on the left; gastric lavage was T.B. positive. On 4.2.47 X-ray (Fig. 7) suggested a satisfactory appearance, but tomograms have shown up small honeycombed areas of disease; some other form of collapse therapy is now indicated.

Grateful acknowledgment is made to Dr. B. W. Anderson, Medical Superintendent, Glenlomond, by Kinross, for his permission to make use of the material on which this work is based, and for providing every facility to make it possible.

REFERENCES

ANDERSON, N. L., and WINN, W. D. (1945): "Pneumoperitoneum and Diaphragmatic Paralysis," *Amer. Rev. Tuberc.*, **52,** 380.

ASLETT, E., and JARMAN, F. T. (1945): "Reaction after Pneumoperitoneum Therapy," *Lancet*, **2,** 304.

BAILEY, W. H. (1948): "Air Embolus in Pneumoperitoneum," *Amer. Rev. Tuberc.*, **57,** 621.

BANYAI, A. L. (1934): "Therapeutic Pneumoperitoneum," *Amer. Rev. Tuberc.*, **29,** 603.

BANYAI, A. L. (1946): "Pneumoperitoneum Treatment." Kimpton, London.

BENATT, A. J. (1946): "Artificial Pneumoperitoneum as an Emergency Measure in Severe Hæmoptysis," *Lancet*, **2.**

BENATT, A. J., and BERG, W. F. (1945): "The Electrocardiogram in Pneumoperitoneum," *Amer. Heart J.*, **30,** 579.

BIRATH, G. (1947): "Pulmonary Function following Pneumothorax," *Amer. Rev. Tuberc.*, **55,** 349.

BRIAN, E. G., and RICEN, E. (1941): "Collapse Therapy of Pulmonary Tuberculosis by Pneumoperitoneum," *Amer. Rev. Tuberc.*, **39,** 391.

Brompton Hospital Reports, 1944 to 1947, Statistics.

CLIFFORD-JONES, E., and MACDONALD, N. (1943): "Pneumoperitoneum in the Collapse Therapy of Pulmonary Tuberculosis," *Tubercle*, **24,** 97.

CORNWALL, V. C., and PARTRIDGE, W. H. (1947): "A Case of Fatal Peritoneal Hæmorrhage complicating Artificial Pneumoperitoneum," *Tubercle*, **28,** 164.

CROW, H. E., and WHELCHEL, F. C. (1945): "Diaphragmatic Paralysis and Pneumoperitoneum," *Amer. Rev. Tuberc.*, **52,** 367.

DROLET, G. H. (1943): "Collapse Therapy," *Amer. Rev. Tuberc.*, **47,** 84.

DRURY, M. I., and DUFFY, J. (1944): "Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis," *Irish J. Med. Sci.*, **00,** 504.

EDWARDS, R. W., and LOGAN, J. (1945): "Pneumoperitoneum in Pulmonary Tuberculosis," *Tubercle*, **26,** 11.

ELLISON, R. T., and TITTLE, C. R. (1943): "Diaphragmatic Paralysis and the Closure of Tuberculous Cavities," *Amer. Rev. Tuberc.*, **47,** 269.

FALES, L. H., and BEAUDET, E. A. (1934): "The Healing of Tuberculous Cavities," *Amer. Rev. Tuberc.*, **30,** 225.

FOWLER, W. O. (1941): "Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis," *Amer. Rev. Tuberc.*, **44,** 474.

FREMMEL, F. (1937): "Phrenicectomy reinforced by Pneumoperitoneum," *Amer. Rev. Tuberc.*, **36,** 488.

GAETAN, L. R. (1939): "Accidental Pneumoperitoneum," *Amer. Rev. Tuberc.*, **39,** 538.

GILMORE, D. J. (1947): "Pneumoperitoneum with Diaphragmatic Paralysis in the Treatment of Pulmonary Tuberculosis," *Dis. of Chest*, **13,** 153.

GRENVILLE-MATHERS, R. (1947): "Cavitation in Pulmonary Tuberculosis," *Tubercle*, **28,** 139.

HAWKINS, F. S., and THOMAS, G. O. (1946): "Basal Pulmonary Tuberculosis with Special Reference to Ætiology and Treatment," *Tubercle*, **27,** 82.

HOWLETT, K. S. (1948): Editorial, *Amer. Rev. Tuberc.*, **58,** 134.

JOANNIDES, M., and SCHLACK, O. C. (1937): "The Use of Phrenic Neurectomy combined with Artificial Pneumoperitoneum for the Collapse of Adherent Lung," *J. Thorac. Surg.* **6,** 218.

KAYNE, G. G., PAGEL, W., and O'SHAUGHNESSY, L. (1943): "Pulmonary Tuberculosis" (2nd Edition). Oxford University Press.

KEERS, R. Y. (1948): "Pneumoperitoneum, its Place in Treatment," *Brit. J. Tuberc.*, **42.**

LOGIE, A. J., WALKER, H. A., and STODDARD, A. R. (1943): *Ann. Int. Med.*, **19,** 685.

MALLICK, S. M. K., MALHOTRA, C. L., and MOHAMMAD, N. (1943): "Pneumoperitoneum in the Treatment of Tuberculosis," *Tubercle*, **24,** 165.

MITCHELL, R. S., HIATT, J. S., MCGAIN, P. P., EASOM, H. F., and THOMAS, C. D. (1947): "Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis," *Amer. Rev. Tuberc.*, **55,** 306.

MORGENSTERN, P., and PINE, I. (1948): "Pulmonary Cavities below the Diaphragm," *Amer. J. Radiology*, **59,** 67.

PINNER, M. (1945): "Pulmonary Tuberculosis in the Adult." Thomas, Springfield, Ill.

POTTER, B. P. (1941): "Results of Collapse Therapy," *Amer. Rev. Tuberc.*, **43,** 184.

RAFFERTY, T. N. (1945): "Artificial Pneumothorax in Pulmonary Tuberculosis." Heinemann, London.

RILANCE, A. B., and WARRING, F. C. (1941): "Pneumoperitoneum supplementing Phrenic Paralysis," *Amer. Rev. Tuberc.*, **44,** 323.

SALKIN, D., CADDEN, A. V., EDSON, R. C. (1943): "The Natural History of Tuberculous Tracheo-bronchitis," *Amer. Rev. Tuberc.*, **47,** 351.
SAMSON, P. C. (1937): "The Treatment and Prognosis in Tuberculous Tracheo-bronchitis," *J. Thorac. Surg.*, **6,** 561.
SHIPMAN, S. J. (1942): "Pneumothorax *v.* Apical Thoracoplasty," *Amer. Rev. Tuberc.*, **45,** 364.
SIMMONDS, F. A. H. (1946): "Air Embolism and Pneumomediastinum in Artificial Pneumoperitoneum," *Lancet*, **1,** 530.
TRIMBLE, H. G., and WARDRIP, B. H. (1937): "Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis," *Amer. Rev. Tuberc.*, **36,** 111.
TRIMBLE, H. G., EATON, J. L., GRENSHAW, G. L., and GOURLEY, L. (1948): "Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis," *Amer. Rev. Tuberc.*, **57,** 433.
TRIMBLE, H. G., EATON, J. L., and MOORE, J. (1939): "Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis," *Amer. Rev. Tuberc.*, **39,** 528.
WARREN, W., HAMMOND, A. E., and TUTTLE, W. M. (1938): "Diagnosis and Treatment of Tuberculous Tracheo-bronchitis," *Amer. Rev. Tuberc.*, **42,** 628.
WATT, J. (1947): "The Complications of Adhesion Section," *Tubercle*, **28,** 158.
WHITEHEAD, G. (1948): "Pneumoperitoneum in the Treatment of Pulmonary Tuberculosis," *Southern Med. J.*, **41,** 681.

RECURRENT NON-TUBERCULOUS SPONTANEOUS PNEUMOTHORAX. A COMPLICATION OF ARTIFICIAL PNEUMOTHORAX TREATMENT OF PULMONARY TUBERCULOSIS

BY M. B. PAUL

From the Liverpool Sanatorium, Frodsham

SPONTANEOUS pneumothorax as a complication of artificial pneumothorax treatment of pulmonary tuberculosis is most commonly due to the rupture of a tuberculous cavity or of a sub-pleural tuberculous focus into the pleural cavity. In both cases a tuberculous effusion is the usual sequel, and Simmonds (1941) believes that the majority of tuberculous effusions complicating an artificial pneumothorax are due to the intra-pleural rupture of a cavity or sub-pleural focus and are preceded by the symptoms and signs of a spontaneous pneumothorax. Another cause of spontaneous pneumothorax is trauma to the lung by a needle, and this may occur at any time during pneumothorax therapy, but is most usual at induction. As healthy lung tissue is involved the perforation in the lung is quickly sealed off without an effusion developing. This type of pneumothorax is seldom recurrent, since the retracting lung effectively seals the perforation. A third group of cases of spontaneous pneumothorax occurring during artificial pneumothorax therapy is so striking that it is worth further consideration. The characteristic features of this group are:

1. Recurrent nature.
2. Absence of effusion.
3. Tension pneumothorax.

Considerable attention has been given to the pathology and treatment of chronic and recurrent spontaneous pneumothorax in the non-tuberculous patient, but very little attention has been paid to the subject as a complication of artificial pneumothorax therapy in pulmonary tuberculosis, and in this

paper the pathology and the management of the condition in eight cases are discussed.

Case Reports

Case 1.—D. T. Male. Aged 26. Bilateral pneumothorax induced. An X-ray film taken after induction of the right artificial pneumothorax showed excessive relaxation of the right lung, suggesting that a spontaneous pneumothorax had occurred, but no adhesions were seen. Four months later a right spontaneous pneumothorax occurred shortly after a refill and air was withdrawn. Two weeks later the spontaneous pneumothorax recurred, again shortly after a refill, and air was again withdrawn on two occasions. In view of the recurrent tendency of the spontaneous pneumothorax the right artificial pneumothorax was abandoned and the lung re-expanded without an effusion developing.

Case 2.—T. W. Male. Aged 15. An X-ray film showed bilateral infiltration with cavitation. An attempt to induce a left artificial pneumothorax failed, but a right artificial pneumothorax was induced to control the disease in that lung. Two months later a spontaneous pneumothorax developed on the day following a refill and air was withdrawn on three occasions. When the spontaneous pneumothorax again recurred water-sealed drainage was established and maintained until the escape ceased. A further recurrence of the spontaneous pneumothorax was again controlled by water-sealed drainage. In spite of the frequent and prolonged escape of air an effusion did not develop. Ascorbic acid (50 mgm. t.d.s.) was given as soon as the drainage was established and was continued for three days. The pneumothorax was maintained without further recurrence for another two months prior to discharge.

Case 3.—G. T. Male. Aged 24. An X-ray film showed bilateral infiltration and cavitation in the upper zones of the lungs. Bilateral artificial pneumothorax was induced; a right spontaneous pneumothorax developed shortly after induction and air was withdrawn. It recurred the following day and air was again withdrawn. A further recurrence took place three days later after a refill and air was once more taken off. Three weeks later the right spontaneous pneumothorax again recurred and air was withdrawn on three occasions in the following few hours. Both pneumothoraces were abandoned in view of the recurrent tendency of the spontaneous pneumothorax. An X-ray film showed the right pneumothorax to be free of adhesions. No effusion developed while the patient was in the sanatorium, but he took his own discharge before the right lung was fully re-expanded.

Case 4.—J. K. Male. Aged 44. An X-ray film showed infiltration and cavitation in both upper zones and emphysema at both bases. Bilateral artificial pneumothorax was induced, and both were complicated by a spontaneous pneumothorax shortly after induction. The right spontaneous pneumothorax recurred the following day, and the left recurred twice following a refill. Both pneumothoraces were complicated by apical adhesions and were abandoned in view of the recurrent nature of the spontaneous pneumothorax and the considerable dyspnœa which the patient experienced. No effusion developed on either side.

Case 5.—O. J. Male. Aged 35. Left artificial pneumothorax induced for control of a spread of disease. Left spontaneous pneumothorax occurred a few hours after induction and air was withdrawn, and air was again withdrawn after a recurrence following a refill two weeks later. The left artificial pneumothorax was abandoned in view of the recurrent nature of the spontaneous

pneumothorax and the failure of the pre-existing right artificial pneumothorax to control the disease in that lung following incomplete pneumonolysis. The left artificial pneumothorax appeared to be free of adhesions and no effusion followed.

CASE 6.—J. M. Male. Aged 27. Right artificial pneumothorax present on admission, but ineffective and complicated by tuberculous empyema. Left artificial pneumothorax induced in an attempt to control the disease in that lung and complicated by a spontaneous pneumothorax which occurred after induction. This recurred twice shortly after refills and air was withdrawn on each occasion. The left artificial pneumothorax was therefore abandoned, although radiologically it was free of adhesions. No effusion developed on the left side.

CASE 7.—J. B. Male. Aged 36. An X-ray film taken on admission showed a good right artificial pneumothorax, and infiltration and probable cavitation in the upper zone of the left lung. A left artificial pneumothorax was induced, but was complicated by apical adhesions. Following a refill three weeks after induction a left spontaneous pneumothorax occurred, and this recurred on each of the three following days, air being withdrawn on seven occasions altogether. The left artificial pneumothorax was maintained without further recurrence of the spontaneous pneumothorax and without an effusion developing.

Pathology

Recurrent non-tuberculous spontaneous pneumothorax of such degree as to be detected clinically is a relatively uncommon complication of artificial pneumothorax therapy. In the last four years approximately 600 artificial pneumothoraces have been induced at the Liverpool Sanatorium and only nine have been complicated by recurrent spontaneous pneumothorax.

Although all the patients in this series were suffering from active tuberculosis at the time, it is reasonable to assume, in view of the absence of effusion, that the cause of the spontaneous pneumothoraces was not the rupture of a tuberculous cavity or focus. The pathology of spontaneous pneumothorax in the apparently healthy person has been widely studied in the last few years, and a number of pathological findings which may be responsible for the condition have been described, and it is reasonable to look among these for the cause of the spontaneous pneumothoraces in this series.

Perry (1939) analysed the causation of spontaneous pneumothorax in eighty-five apparently healthy persons seen at the London Hospital and concluded that, although spontaneous pneumothorax may occur as a complication of known pulmonary tuberculosis, yet pulmonary tuberculosis is not a cause of spontaneous pneumothorax in the apparently healthy, as shown by the failure to develop a hydro- or pyo-pneumothorax. Perry dismissed rupture of adhesions as a cause of spontaneous pneumothorax and believed that this was more likely to give rise to a hæmo-pneumothorax, and concluded that benign spontaneous pneumothorax must usually arise from the rupture of a sub-pleural air vesicle, either of emphysematous, scar tissue, or congenital origin.

Brock (1948) has also made an exhaustive study of the cause of recurrent and chronic spontaneous pneumothorax in a series of seventy-one cases, and has described several pathological findings in the lung which, together with a

common mechanical factor, could be held responsible for the condition. Several of the causative conditions in Brock's series of cases can be dismissed as probable etiological factors in this series, as, for example, large solitary cysts and polycystic disease of the lung which would contra-indicate artificial pneumothorax treatment. Generalised emphysema also would make a patient unsuitable for artificial pneumothorax treatment, although this may have been a factor in Case 4. It seems, therefore, most probable that the causes of the spontaneous pneumothoraces in this series will be found among the following conditions.

1. Apical Emphysematous Bullæ

Small apical emphysematous bullæ are not uncommonly seen on X-ray films of the chest, and their presence is generally accepted as evidence of the existence of an old tuberculous lesion. Rupture of these bullæ has been demonstrated at autopsy to be the cause of spontaneous pneumothorax, and this may occur at rest, or may be precipitated by violent exercise or straining or by a severe bout of coughing. Meade and Blades (1949) reported nineteen cases of chronic and recurrent spontaneous pneumothorax and found that the cause of the pneumothorax in ten of the cases was rupture of or leakage from emphysematous blebs. Emyhysematous bullæ have been observed at thoracoscopy prior to division of adhesions in several cases undergoing artificial pneumothorax treatment for pulmonary tuberculosis, and have been noted to inflate and deflate with respiration.

2. Apical Scars

Apical scars may be seen radiologically without definite bullæ being demonstrable, and in these cases Brock assumes that the escape of air has come from a smaller emphysematous area or from minute bullæ secondary to scar tissue. Here one would expect the development of the spontaneous pneumothorax to be a slow process and the accumulation of sufficient air in the pleural cavity to take some time. This has, in fact, been observed clinically and a history of gradually increasing dyspnœa obtained.

3. Rupture of Adhesions

Rupture of adhesions during the course of artificial pneumothorax treatment undoubtedly does occur, although it is much more rare than it used to be when attempts were made to stretch adhesions by the use of high intrapleural pressures. When this happens the break is at the weakest point, which is usually at the junction of the adhesion and the lung, and a tuberculous effusion usually results. This is not, however, invariable, and Cutler (1948) has reported a case of tension pneumothorax due to a tear in the lung at the base of an adhesion. The absence of tuberculous effusion must be explained by supposing that there was no active tuberculous disease under the adhesion. Meade and Blades (1949) also report a case of chronic spontaneous pneumothorax due to a band adhesion maintaining a small bronchopleural fistula, which closed after the adhesion was divided by internal pneumonolysis.

The onset of spontaneous pneumothorax was observed to be closely related in time to the giving of a refill in all the cases in this series, and to the size of the refill in some; thus when only a small refill sufficient to maintain a shallow pneumothorax was given the complication did not occur, but when it was attempted to increase the relaxation of the lung by giving a large refill a spontaneous pneumothorax developed. From these observations it is tempting to deduce that the most frequent cause of the complication was a tear in the lung at the base of an adhesion which was put on the stretch following the refill, and that a subsequent bout of coughing or straining caused the actual tear. An alternative explanation, especially for those cases in which the pneumothorax appeared to be free of adhesions, is that the operative mechanism is similar to that which is considered to be responsible for the formation of a tension cavity, only in this case an emphysematous bulla takes the place of a tuberculous cavity. This mechanism can be held to explain the development of a spontaneous pneumothorax after a large refill, for the diminution in the volume of the lung will also cause a narrowing of the bronchioles themselves, since they contain no cartilage in their wall and this could produce a check valve in the bronchiole proximal to a bulla. Another possible factor in the development of the complication in this series was the previous existence of an artificial pneumothorax, and it is reasonable to suppose that a spontaneous pneumothorax is more likely to develop in cases in which the visceral pleura is unsupported than when it is in contact with the chest wall.

The most informative diagnostic procedure is thoracoscopy, which may reveal the presence of an actual tear or rent in the lung under an overlying adhesion, the presence of emphysematous bullæ or of multiple cysts of the lung, or the information obtained may be negative. Unfortunately thoracoscopy was not carried out on any of the cases in this series, as it was not possible to perform the operation at short notice, and consequently there is no direct evidence as to the cause of the complication in any case. Of the nine artificial pneumothoraces in which this complication occurred four appeared to be entirely free of adhesions radiologically, and it is probable that rupture of an emphysematous bulla was the causative factor in these cases. Case 4 was complicated by emphysema as well as pleural adhesions and this may have been a contributory factor in the development of the complication. In the remaining cases adhesions were present to a variable degree and rupture of the pulmonary attachment of these may have been an etiological factor.

Management

The problem of management of these cases is twofold. Firstly, there is the immediate treatment of the tension pneumothorax, and secondly the treatment of the pulmonary tuberculosis for which the artificial pneumothorax was originally induced. The treatment of the tension pneumothorax is a matter of urgency, but it is also necessary to bear in mind the more remote treatment of the tuberculosis.

The tension pneumothorax may be controlled by withdrawing air on one or more occasions, but in some cases it may be necessary to insert a small cannula into the pleural cavity and to set up continuous water-sealed drainage.

This was required in two of the cases in this series. Ascorbic acid (50 mgm. t.d.s.) was given to both of these cases in the hope that it would facilitate healing of the perforation.

The decision as to the best way of treating the original pulmonary disease can be made only after consideration of several factors. In some cases, depending on the extent of the disease, the state of the contra-lateral lung, and the prospects of alternative methods of collapse therapy, it may be advisable to abandon artificial pneumothorax therapy at once. In other cases, particularly those in which the disease is bilateral, artificial pneumothorax may seem to hold the best prospects of controlling the disease, but if it is decided to maintain the pneumothorax thoracoscopy is indicated, as this may reveal the cause of the complication and will be needed to demonstrate the presence or absence of pleural adhesions which can be divided at the same time.

The risks of a recurrence of the spontaneous pneumothorax are considerable, and only in exceptional cases should the pneumothorax be maintained without steps being taken to diminish this risk. In cases due to a tear in the lung at the base of an adhesion the risk can be removed by dividing them. In those cases in which the complication occurs in a pneumothorax free of adhesions the risks of recurrence can be reduced by performing a partial pleurodesis, but only when the disease is confined to the apex of the lung and the pneumothorax is highly selective.

Artificial Pleurodesis

Artificial partial pleurodesis was performed once in this series (Case 8). The pneumothorax appeared to be free of adhesions and selective, and the disease was confined to the upper zone of the lung. Even after partial pleurodesis it is possible for a spontaneous pneumothorax to recur as the site of the leak may be in that part of the lung which it is desired to keep collapsed, but should this occur, the possibility of a total pneumothorax is excluded by the fixation of the greater part of the lung to the chest wall. The therapeutic possibilities of artificial pleural effusions were described by Maurer (1937), who treated cases of bilateral apical pulmonary tuberculosis by artificial pneumothorax and fixed the healthy lower zones of the lungs to the chest wall by this means, thus ensuring the maximum respiratory function in the healthy zones of the lung. He used as an irritant a 50 per cent. solution of glucose. Other workers have used a variety of irritants, such as insufflation of powdered talc (iodised or plain), injections of a suspension of talc, injections of venous blood, iodised poppy-seed oil, gomenol in olive oil, and solutions of silver nitrate in varying strengths. Injection of all these substances causes some degree of pain and febrile reaction, and in most cases a transient effusion develops. If the two pleural surfaces are in apposition at the time they will adhere and, provided they are not separated in the early stages by too large refills being given, they will be firmly united in a few weeks' time. One of the dangers of artificial effusion is extension of the obliterative process further than was intended, and it calls for fine judgment in assessing the need for refills, and striking a balance between allowing the obliteration to rise too high and between producing premature separation of the pleural surfaces.

CASE 8.—G. M. Female. Aged 21. An X-ray film taken on admission showed infiltration and miliary deposits in the upper zones of both lungs. Sputum was positive. A right artificial pneumothorax was induced and X-ray showed this to be free of adhesions. A few hours after induction a spontaneous pneumothorax occurred and air was withdrawn. Further recurrences took place on each of the four succeeding days and air was withdrawn on all of these occasions. Eventually water-sealed drainage was instituted and maintained for forty-eight hours. No further recurrence took place, but before attempting to collapse the left lung it was decided to produce a partial pleurodesis on the right side to reduce the risk of recurrence. This was done by injecting five minims of 10 per cent. silver nitrate into the pleural cavity when the lower zone of the right lung was in contact with the chest wall. The injection of the silver nitrate caused a sharp febrile reaction which lasted for six days. Four days after the injection screening showed a considerable effusion, which was aspirated, 33 oz. of clear fluid being withdrawn and replaced by 100 c.c. of air. A subsequent X-ray film showed the lower half of the lung in contact with the chest wall below an apical pneumothorax. In order to test the firmness of the pleural symphysis the pneumothorax was refilled to a positive pressure about eight weeks later, and an X-ray taken the following day showed that the lower two-thirds of the lung were firmly adherent to the chest wall. A left artificial pneumothorax was then induced. The area of adhesion between the two pleural surfaces did not increase and the right apical pneumothorax was maintained with small refills of 80 to 100 c.c. of air at weekly intervals. No further recurrence of the spontaneous pneumothorax occurred while the patient was in the sanatorium.

Summary

1. Spontaneous pneumothorax complicating the artificial pneumothorax treatment of pulmonary tuberculosis is most frequently due to the rupture of a sub-pleural tuberculous cavity or focus, and a tuberculous effusion is the usual sequel.

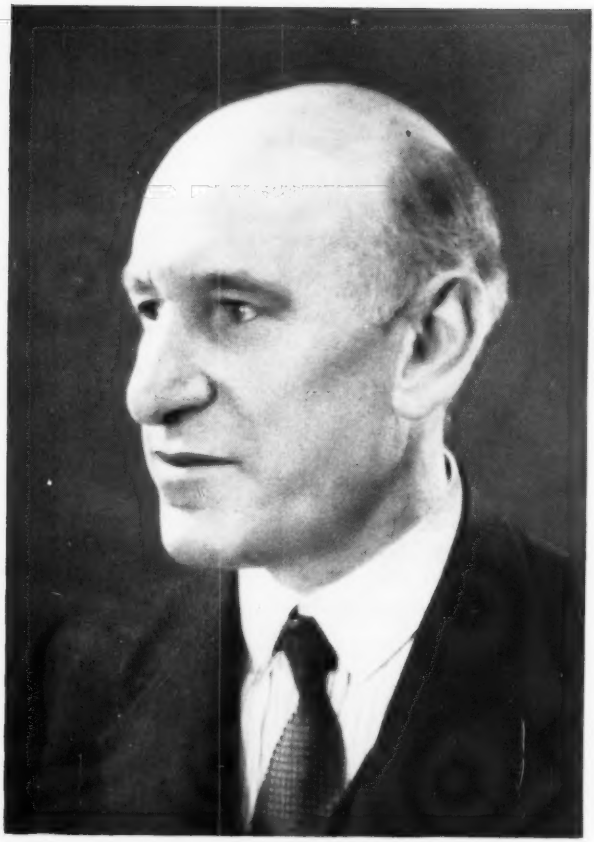
2. In cases characterised by (*a*) recurrent tendency, (*b*) absence of effusion, and (*c*) tension pneumothorax, it is suggested that the spontaneous pneumothorax is due to rupture of an emphysematous bulla or apical scar or to a tear in the lung at the base of an adhesion.

3. The management of the complication is discussed and the treatment of a case by artificial pleurodesis is described.

I wish to thank Dr. G. S. Erwin, Medical Superintendent of the Liverpool Sanatorium, for permission to publish these cases, and for his criticism and advice.

REFERENCES

BROCK, R. C. (1948): *Thorax*, **3**, 88.
CUTLER, J. W. (1948): *Journ. Thor. Surg.*, **17**, 374.
MAURER, G. (1937): *Brit. J. Tuberc.*, **31**, 58.
MEADE, R. H., and BLADES, B. H. (1949): *Amer. Rev. Tuberc.*, **60**, 683.
PERRY, K. M. A. (1939): *Quart. Journ. Med.*, **8**, 29, 1.
SIMMONDS, F. A. H. (1941): *Tubercle*, **22**, 183.



SYDNEY VERE PEARSON
1873—1950

To face page 51

IN MEMORIAM

SYDNEY VERE PEARSON

M.A., M.D.Cantab., M.R.C.P.Lond.

THE death of Dr. Vere Pearson, late Superintendent of the Mundesley Sanatorium, has taken from us yet another of the real physicians of the old school, to whose strong personality and experienced outlook on the problems of human disease and suffering the fine tradition of British medicine owes so great a debt. The details of his professional career have been set forth in other medical journals and need no repetition in these pages. I have been asked to write a personal appreciation of the man as I saw him, and it is with no little pride in his memory and admiration for his professional methods that I recall those impressions of his character which stand out in my mind as the landmarks by which he should be remembered.

The influences of his earlier life were varied, and combined in wondrous wise to form that composite character of which so many facets glittered in the general picture familiar to those who knew him. It needs but little imagination to analyse the influences which must have moulded the pattern of his life and work. The son of a Liverpool Congregational minister, in whose home were doubtless sown the first seeds of that sense of reverence and that strength of purpose which were among his distinguishing hall-marks, Pearson became a pupil of the Manchester Grammar School, that great institution in which, though justly famous for its standards of classical education, character counted for as much as scholarship. Both of these were conspicuous in his university life. An exhibitioner of his college, he graduated with honours in the Natural Science Tripos, and later on received his blue, rowing in the Cambridge boat in 1898.

One could hardly, perhaps, suggest a better combination of experiences to form the background of a medical student destined to make his mark among the men of his profession. That blending of first-class technical knowledge with an appreciation of the human side of affairs, which made him the great physician that he was, can be traced in the development of his school life, in his academic training and in his early post-graduate medical career. The enforced interruption by illness of his programme at a critical stage not only determined the particular avenue along which his medical activities were ultimately to run, but must have influenced profoundly his mode of approach to the problems to which his life was to be devoted. Those who were his colleagues at Mundesley and who learned so much from his quiet teaching and example will recognise this as a fundamental truth. His prescience in tuberculosis work was the result of a long training in general medicine, experience on the honorary staff of a children's hospital, a devotion alike to the experimental and to the clinical aspects of chest disease, and, above all, a sympathetic knowledge of mankind with which Nature and environment had combined to endow him.

Pearson's hobbies were numerous, but all of them bespoke his wide cultural outlook. The briefest sketch of him would be incomplete without a reference

to his heartfelt interest in politics and his attachment to the Independent Labour Party. This was well known, not only to his colleagues but also to his patients, and it was sometimes jestingly, though not unkindly, said of him that he was more interested in attending the local political meetings than in attending to the Sanatorium. The passionate desire for justice, as he saw it, was without doubt the basis of his political views, and never was he accused, even by the bitterest political opponents, of any attempt to force these upon others, though on such matters he felt very deeply and with all the innate sincerity which was the keynote of his attitude to his patients. To the latter he was a figure of great presence, who could and did inspire no little awe, though it was probably at times when they were most afraid of him that his own understanding of their difficulties and sympathy with their griefs were most active, albeit inarticulate. To his colleagues at Mundesley he became more and more a source of strength as time passed and their understanding of him deepened. Not long before his retirement I was speaking to one of them and I inquired if Pearson was still taking a very active part in the sanatorium work; the answer was, " No; he has more or less retired, but about every three weeks or so he suddenly comes round and tells us we are doing it all wrong !" From the joy with which this remark was made and from the manner of its utterance I realised what tribute had been paid to the physician of whom it was said.

Two of the finest tuberculosis experts I have ever known were Wingfield of Frimley and Vere Pearson of Mundesley. Although these two men were vastly dissimilar to outward view, I have often felt that there was something in which they were alike and which was the secret of their greatness. It is difficult to define, but I think it was the combination of science and the humanities. This is productive of a rare understanding, which, as in both these men, is often cloaked under an outward appearance of severity that really betokens an innate shyness and an intense human sympathy. Of Vere Pearson I believe this to be true in a high degree, and I join with many friends in the medical profession in deploring the loss occasioned by his death.

MAURICE DAVIDSON.

REVIEWS OF BOOKS

Photo-radiography in Search of Tuberculosis. By David W. Zachs. Pp. xii+297, with 274 illustrations. Price 40s. net. London: Baillière, Tindall and Cox.

In his book *Photo-radiography in Search of Tuberculosis*, Dr. Zachs gives a sympathetic account of the problems, social, technical and administrative, which are created in the attempt to search out pulmonary tuberculosis by miniature radiography, and shows how these problems are dealt with in the U.S.A. He covers every aspect of the complex process of miniature radiography, showing its place in the Tuberculosis Service and giving to the many details of his work a cohesion which makes reading easy.

A comprehensive account of the unit, of its facilities (with explanatory photographs), of the personnel and their duties and protection from radiation, of the dangers of overloading the X-ray tube, and the many factors which help to make direction of the unit easy.

In planning the survey, the preliminary education of the available personnel, the relationship between management and factory hand, publicity and management are discussed. The organisation of assembly of personnel in town and country is described, schedules of work are detailed, and a meticulous account of how each person comes to be X-rayed and is informed of its result is given. Investigation of suspects, the problem of disposal and the integration of the work with the general practitioner and chest physician is described.

On the technical aspect of photo-radiography, those who have experience with the matter will agree that he deals with the essentials clearly and briefly whilst giving the information which is needed.

A brief account of how miniature films should be read is given, the optimum conditions for reading them are detailed, and a short chapter is devoted to the diagnostic pitfalls and diagnosis of early pulmonary tuberculosis.

His atlas of 270 reproductions is well annotated. They are mainly devoted to the findings of mass miniature radiography and concentrated on the appearance of pulmonary tuberculosis and of artefacts, with examples of other conditions that may occur.

This book is well written; writing a balanced account of so involved a process as mass radiography is difficult, but Dr. Zachs has been successful. He has written a well-balanced and integrated account which should be read by all critics of mass miniature radiography and which can be read as a book of reference by clinicians who use this method of approach towards the eradication of tuberculosis of the lungs. He has integrated technical knowledge with social science and public health to provide a useful account of a weapon with which we can fight against this disease.

D. K. M. T.

The Middlesex Hospital. By Hilary St. George Saunders. London: Max Parrish. Price 8s. 6d.

It was a happy thought of the authorities to request the well-known librarian of the House of Commons to write the history of the Middlesex Hospital up to the "appointed day," July 5, 1948, when the "voluntary hospital system" virtually ceased to exist. It is well that the general public (and also future members of the medical profession) should have preserved for them the atmosphere which permeated such institutions in the past.

The story begins in 1756, ten years after the hospital was founded, and at that time the weekly board meetings were held at the "Bear and Rummer,"

assisted by a lunch of bread and cheese and unlimited supplies of ale. The secretary to the governors had been instructed to obtain a contract with Covent Garden for the supply of millepedes (otherwise known as wood lice). The expressed juice of fifty living millepedes was said to cure obstinate jaundices. Nearly 200 years later, in 1938, one of the new sulphonamide drugs was given its clinical trials at the Middlesex Hospital.

In the years between these two types of treatment the hospital was the training ground of many of the well-known clinicians and also of workers in radiotherapy who built up the world-wide fame of British medicine.

This book is strongly recommended for a place on the shelves of all who are interested in the social, as well as the medical, life of the last 200 years. It makes fascinating reading.

G. S.

Tuberculosis Nursing. By JESSIE C. EYRE, S.R.N., T.A.(Hons.), Senior Sister Tutor, St. Helier Hospital, Carshalton. London: H. K. Lewis. Pp. xii+292. 102 illustrations. Price 21s. net.

In this book Miss Eyre has succeeded in setting out the essentials of tuberculosis nursing in a most attractive and readable form. Perhaps its outstanding features are the accuracy of the statistical data it contains, the excellent way in which the pathology of tuberculosis is dealt with, and the high standard of the illustrations. Faults can be found within its pages—one or two of the diagrammatic interpretations of the X-ray plates are not quite accurate, and syphilis of the lung is so rare and controversial a subject that it should be left out of a book of this description. In addition, the section on bronchography might well include an account of other methods than the crico-thyroid route and at the same time set down clearly the indications for the procedure, lest it give the student nurse the false impression that it is an investigation commonly carried out in pulmonary tuberculosis. But these are minor faults and the author is to be congratulated on having written one of the best books of its kind on the subject.

R. P. K. C.

AMONG others, the following volumes have been received:

The Tuberculous Process. By Alfred Leitch. Published by John Wright, Bristol. 1949. Price 12s. 6d.

Tuberkuloselexikon für Ärzte und Behörden. By W. Roloff. Published by Georg Thieme, Stuttgart. 1949. Price DM. 14.80.

Aktuelle Probleme der Pathologie und Therapie. By H. Holthusen. Published by Georg Thieme, Stuttgart. 1949. Price DM. 29.

Neuere Tuberkuloseforschung—I. By R. Griesbach. Published by Georg Thieme, Stuttgart. 1949. Price DM. 6.